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Original Articles.

EXTENSIVE FIBROSIS OF THE LEFT VENTRICLE IN A CHILD.

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THE patient, W. C—, aged 10 years, a boy of Russian Jewish parentage, was admitted to hospital on March the 8th, 1916, with a history of recent melæna. He was rather delicate-looking and had considerable enlargement of the heart. Crepitation was at first present at the base of the left lung behind, but could no longer be heard by March the 20th. In the hospital he frequently had more or less melæna, and occasionally there was a little fever. Sometimes he complained of headache. The urine was free from albumin and sugar. There was a history of his having had an illness, accompanied by the passage of blood *per rectum*, two years previously.

On April the 7th severe hæmorrhage occurred from the bowel, and, as a result, the boy became collapsed and blanched from the loss of blood. In the afternoon of that day no pulse could be felt at the wrists, and an intravenous injection of physiological salt solution was made, with good result. During the following days a continuous "drop-enema" of physiological saline solution was frequently made use of, and the patient's condition gradually improved. Calcium lactate was given by the mouth. There was practically never any fever. Apart from the loss of blood *per rectum*, the patient had sometimes had, since the illness of two

years ago, and during the first part of the present illness, slight hæmatemesis and slight hæmoptysis.

On April the 14th I noted that the fæces were quite free from blood, even by the modified guaiac test. No fever whatever was charted after April the 16th, and the patient left the hospital on June the 13th in apparently satisfactory condition, except for the enlarged heart. Temporarily (May the 5th) the fæces again gave a positive reaction for blood. Early in June I noted that the boy was gaining weight and seemed well and happy. The pulse was, however, generally between 100 and 120 per minute, and the respiration between 26 and 28. The urine contained no albumin or sugar. The cardiac impulse was forcible, and the apex beat was in the sixth left intercostal space in the anterior axillary line; no murmur of any kind could be detected.

On June the 20th the boy looked pale and vomited, and he was readmitted. The urine then showed a trace of albumin, but no tube-casts. The fæces contained no blood. He soon appeared better, and was allowed to leave the hospital on June the 27th.

On June the 30th he was readmitted, complaining of a feeling of painful "stiffness" in the legs and abdomen. In the hospital after this there was sometimes slight fever. The urine at first contained albumin, but was afterwards reported free from albumin and sugar; no blood could be detected in the fæces. On July the 4th the feeling of "stiffness" seemed to be passing off. However, on July the 5th, at 8 a.m., the boy began to complain of violent pains in the abdomen, and when I saw him later on in the morning he was suffering also from pains in the lower extremities, chiefly in the right foot and leg. The right foot was at that time very pale and cooler to the touch than the left leg, which showed a "livedo-like" mottling. By 3 p.m. the pallor of the right foot had extended up the leg almost to the groin. Embolism was, of course, thought of, but owing to the patient's collapsed condition the pulse-test could not be employed, as no pulse could be felt in either lower extremity nor at either wrist. Since the commencement of the final attack—that is to say, since about 8 a.m.—there had been occasional vomiting, but not of blood. Death occurred at 3.30 p.m.

Necropsy and Microscopical Examination.

The heart weighed $12\frac{1}{2}$ oz. and was both hypertrophied and dilated. The myocardium of a large portion of the outer wall of the left ventricle had undergone a fibrous change, having been converted

into hard, white, scar-like tissue. In the cavity of the left ventricle was an adherent, rather soft, organised ante-mortem thrombus, firmly planted on (as if "growing from") the endocardium near the apex. There was no valvular disease. The great trunks of the coronary arteries were not stenosed.

There was considerable disease of the abdominal aorta. Microscopic examination of this part of the aorta showed great thickening and atheromatous changes in the inner coat (*intima*),

FIG. 1.—Part of the wall of the left ventricle (magnification × 7). It shows the extreme degree of the fibrosis and the distribution of well-preserved muscle-fibres left around the minute blood-vessels; also two arterioles partially blocked by endarteritis proliferans.

together with some small-cell infiltration about the *vasa vasorum* of the muscular-elastic coat (*tunica media*) and the outer coat (*adventitia*). At one spot, where there was an atheromatous deposit, the muscular-elastic coat was thin, and bulging outwards, to form a kind of "miliary aneurysm."

Microscopic sections of the fibrotic wall of the left ventricle (see Figs. 1 and 2) gave a very striking picture. The small arteries were extremely affected by a process of endarteritis proliferans (obliterans), so that their channels were greatly obstructed, but not

altogether obliterated (see Fig. 3). The fibrous tissue which constituted the greater portion of this part of the cardiac wall contained numerous dilated thin-walled blood-vessels (capillaries), suggesting that, owing to the obstructive disease in the small arteries, the nutrition had perforce to be maintained, as far as it could be, through

Fig. 2.—Part of Fig. 1 under higher magnification (× 17), showing the distribution of well-preserved muscle-fibres around minute blood-vessels.

compensatory dilatation of the capillaries. There was very little small-cell infiltration to point to any (recent) inflammatory activity. The surviving (well-preserved) cardiac muscle-fibres tended in their arrangement to be grouped around small blood-vessels, mostly capillaries. This is well shown in Fig. 2, and suggests that the change in the diseased part of the heart-wall is largely one of "fibrous substitution," or "replacement-fibrosis," of ischæmic origin. In other

words, it seems as if the muscle-fibres—that is to say, the "noble" or more highly differentiated histological elements of the cardiac wall—had given place to the growth of the less highly differentiated and more easily nourished ("hardier") connective tissue elements ("fibrous substitution" in the myocardial framework), excepting in the immediate neighbourhood of the thin-walled blood-vessels, where the nutritional requirements of the "nobler" elements could still be satisfied. In regard to the life of the whole organism the "conservative" nature of a replacement-fibrosis of this kind is evident.

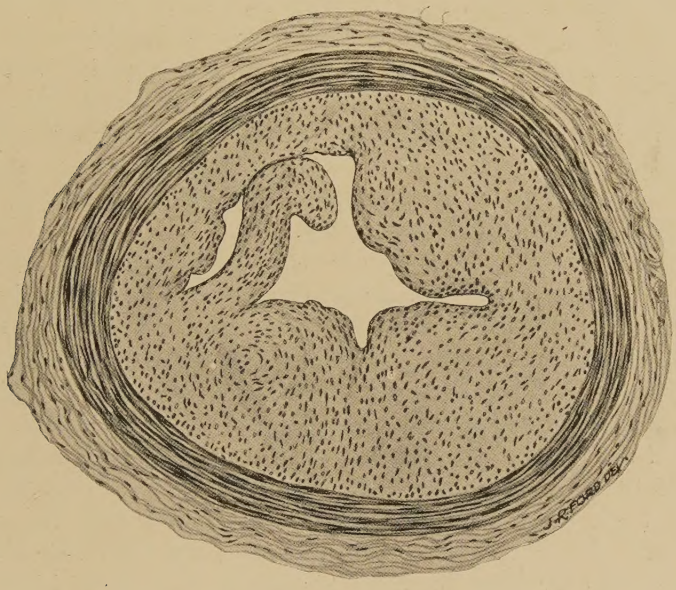

Fig. 3.—An arteriole in the wall of the left ventricle (magnification × 57) whose lumen is greatly stenosed by arteritis proliferans.

A fibrous patch in the wall of the heart, such as I have described in the present case, generally prevents rupture, and often to some extent prevents local bulging (though ultimately a cardiac aneurysm is not unlikely to develop), whilst the rest of the myocardium is able to carry on the heart's work sufficiently well (for a considerable time probably) to maintain the life, and permit moderate activity, of the whole organism.

As to the remaining post-mortem findings I will be as short as possible. The lungs showed some catarrhal changes, and there were old pleuritic adhesions between the left lower lobe and the diaphragm. There was no peritonitis. The right common iliac

artery was occluded at its bifurcation by recent embolism. Nothing was found in the stomach and intestines to account for the attacks of melæna, etc., and no embolism of the mesenteric arteries was detected. The kidneys, which together weighed 9 oz., showed many embolic infarcts of various ages. The left suprarenal gland appeared macroscopically and microscopically normal, but the right suprarenal gland was enlarged to the size of a large chestnut and weighed $\frac{1}{2}$ oz. A minute portion of the right gland consisted of normal suprarenal gland substance, but almost the whole of it (microscopic examination) had undergone a diffuse fibrosis, which Mr. S. G. Shattock compared to that seen in congenital syphilitic livers in cases of so-called intercellular (less correctly, pericellular) cirrhosis in infants. Dr. H. Schmidt suggested that in the present case the degenerative change, whatever its nature was, had occurred, not in a normal suprarenal gland, but in a cortical adenoma of the gland, suprarenal adenomata being especially liable to undergo degenerative changes. At one part of the (right suprarenal) gland a recently thrombosed vein was noted.

The liver (weight, 28 oz.) was examined microscopically, but showed nothing pathological, except chronic passive congestion. A small caseous lymphatic gland was found near the liver. The spleen, the pancreas, the testes, and the remains of the thymus gland were not diseased. Incidentally, it may be mentioned that a little submucous nodule, of the size of a small pea, was found at about the middle of the greater curvature of the stomach, and microscopically appeared to be a misplaced fragment ("rest") of pancreatic tissue. Though fragments or "rests" of "aberrant pancreatic tissue" are well known to occur in the small intestine, I am not aware that any of them have ever been described as having been found in this part of the stomach.

Dr. H. Schmidt examined specially stained microscopic sections of both suprarenal glands, the thymus, and the liver, for spirochætes, but with negative result.

The post-mortem examination showed, as stated, that the boy had an adherent organised thrombus in the left ventricle of his heart, which had been giving off emboli. This cardiac thrombosis had developed secondarily to an old fibrous transformation in the heart-wall, which, there can scarcely be any doubt, was of congenital (inherited) syphilitic origin. The endarteritis proliferans of the little cardiac arteries seen in Figs. 1 and 3 is by itself almost sufficient proof of the syphilitic nature of the change in the diseased portion of the heart-wall. The patient's blood-serum was not

examined for Wassermann's reaction, but the Wassermann reaction was found to be negative in his father and in his supposed mother. It transpired, however, that the father's present wife, whom I had supposed to be the mother of the boy, was not the mother. His real mother had been a woman with whom the patient's father associated when doing military service in the Russian army. The present wife of the patient's father had had by him no children, but two or three miscarriages.

In regard to the special distribution of the still preserved muscle-fibres in the affected portion of the heart-wall I should like to draw attention to Figs. 1 and 2 in Dr. A. Weber's 'Thèse de Paris' (Steinheil, Paris, 1887), entitled "L'Artériosclérose du Cœur (Scléroses du Myocarde)," where very similar changes in the myocardium are illustrated. Similar appearances were likewise illustrated by Prof. H. Huchard in his 'Traité Clinique des Maladies du Cœur et des Vaisseaux,' second edition, Paris, 1893, p. 174, fig. 20; p. 177, fig. 21, plate ii, fig. 3. About 1893, when I was at Paris, Prof. H. Huchard and Dr. A. Weber showed me a microscopic section from a portion of fibrosed cardiac wall, showing muscular changes practically identical with those in the present case.

In conclusion, I have to thank Dr. H. Schmidt for his great assistance, especially in regard to the pathological examination of the case, and Mr. S. G. Shattock for kindly looking through the microscopic specimens with me and for help in regard to the illustrations.

THE TREATMENT OF RICKETS, BASED ON A NEW THEORY AS TO THE PATHOGENESIS, WITH AN ILLUSTRATIVE CASE.*

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The case of rickets which I show this afternoon is brought before the Section for the purpose of illustrating the good results which can follow when the treatment of the condition is based in accordance with certain principles which I venture briefly to outline.

In accordance with the view of the pathogenesis of rickets which

* A paper read before the Section for the Study of Disease in Children of the Royal Society of Medicine on May the 26th, 1916.

I suggest, the symptoms usually ascribed to the condition may be regarded as representing the results of the calling into play by the victims of the disease of certain protective mechanisms. The troubles from which they protect the organism in the case of rickets are those which arise in connection with the disposal of an excess of nutritive material.

In order that an individual may remain in health, sufficient food material must be provided for the maintenance of the normal temperature, for the performance of such mechanical work as may be required for the ordinary purposes of life, for the growth and maintenance of the tissues, and also for the elaboration of secretions.

If the total calorie value of the food exceeds these requirements, it follows that the excess must be disposed of in some manner, which, inasmuch as it does not subserve any required or necessary purpose, may be regarded as pathological.

Recognising the strictly conservative character of the organic reactions in the animal body, we may assume that under given circumstances the method of disposal adopted will be that which will inflict the least possible degree of injury on the organism as a whole.

By what methods can excess of food be disposed of? There are many alternative expedients, of which the following are examples. Classified in order of physiological advantage we may assume that the most economical expedient is to store up the excess in the form of a food reserve—for instance, as glycogen or fat. The limits of storage, especially as far as nitrogen is concerned, are, however, under all conditions extremely circumscribed, and occasionally quite negligible.

A second method is that of combustion or oxidation to the normal end-products carbonic acid gas, urea, and water. This method is economical in that it entails little strain on the mechanisms of elimination. On the other hand, it implies a waste of energy in certain other directions, as, for instance, in the dissipation of the heat produced and in the supply of the necessary oxygen.

A third alternative depends on the short-circuiting of the oxidation processes, in fact on the production of incompletely burnt-up products of combustion. This method has the advantage of saving oxygen and of limiting heat production, but the disadvantage of flooding the blood with acid bodies of large molecular size. I mean such bodies as lactic, oxalic, uric, glycuronic, di-acetic, beta-oxybutyric, and certain other organic acids. Such acid bodies are not allowed to remain in the blood without neutralisation, for acid

reactions are fatal to cell activities; hence they are neutralised almost before they are formed at the expense of alkalies or basic elements derived from the floating reserves or from the tissues themselves. After neutralisation they are, of course, removed from the system by the ordinary processes of elimination. In this way the reserves of carbonates in the blood are drawn upon, ammonia is extracted from protein sources, while calcium, sodium, magnesium, and iron are withdrawn or withheld from various important organic combinations.

The injuries inflicted on the organism by this method of disposal are those which are commonly covered by the comprehensive term "acidosis."

The symptoms by which we recognise an acidosis are as follows: In serious cases we observe "acyanotic hyperpnœa," due to the depletion of carbonic acid carriers and concomitant stimulation of the respiratory centres; there may be serious cyclic vomiting, possibly due to an associated stimulation of the pneumogastric centres, as well as a great variety of nervous symptoms dependent on analogous causes. Among the more chronic results are disintegration of the red blood corpuscles, interference with the oxygen-combining powers of hæmoglobin, and demineralisation of the osseous tissues. One of the most interesting results of the hæmolytic disturbances thus set up is the compensatory activity of the blood-forming organs, the hæmogenetic functions of the red marrow being stimulated in corresponding degree. The external evidences of this functional and compensatory activity are the enlarged epiphyses of long bones so common in rickety conditions.

The resources at the disposal of the organism for dealing with excess of nutritive material are by no means exhausted by the methods already enumerated; but they represent the most important, and include among their results the symptoms which are generally regarded as making up the clinical picture of rickets.

In any particular case of rickets it is impossible to predict what precise method or combination of methods will be adopted to dispose of the excess, and herein lies the protein character of the symptoms of the disease. But however much they may be disguised or obscured, or however incompletely we may be able to follow the intermediate stages, we may feel perfectly confident that in the final issue the metabolic processes will follow the rule of physiological expediency, obey the laws of the conservation of energy, and conform to the economic necessities of supply and demand. The fulfilment of these laws and conditions must result in the manifestation of the

kind of symptoms I have described, and which make up the clinical picture of rickets.

If this theory of rickets is true, as I firmly believe it is, it is necessary to prove that in every case of rickets there is some excess of food, otherwise the acidosis and the other manifestations of the disease cannot be explained on the lines suggested. If by "excess" we simply mean "excess" in the popular interpretation of the term, and as estimated by the usual arbitrary standards, it must be admitted that the theory falls to the ground. But if by the term we mean "relative excess," that is to say a redundancy over and above that which is required for the physiological purposes, then I submit that the theory holds good in every detail. I mention this because rickets is generally included among the deficiency diseases.

The physiological food demands of many infants and children are negligible. If they are kept in hot, stuffy rooms, if they are wrapped up in a multiplicity of clothes, if they are seldom taken out of doors, and if they are given no opportunities for muscular exercise, they will create no demand for food, and consequently any dietary, however small, may be *relatively excessive*, and, if excessive, must be disposed of by one of the protective methods already described. These are the usual conditions which surround the victims of rickets. Under such circumstances we could predict with complete confidence that the child would, if he could, lay up stores of glycogen and fat and become obese; that he would show evidence first of excessive combustion by sweating and vascular dilatation of the superficial capillaries of the face and other exposed parts, and possibly by disturbances of the heat regulating centres, and then of sub-oxidation with the symptoms of an acidosis, with enlarged epiphyses and demineralisation of bone; and finally of acyanotic hyperpnœa with other serious nervous manifestations.

I have for many years treated all cases of rickets on the assumption that this is the true pathogenesis of the disease; but I have selected this particular case to show because it has responded peculiarly well to the expedients which such an assumption suggests.

The following is a short history of the case. W. H—, a girl, aged 2 years, admitted to the Queen's Hospital for Children, February the 28th, 1916, for spinal disease, supposed to be tuberculous.

Family history.—Mother married twenty-four years, fifteen pregnancies, four miscarriages. Ten children alive and healthy. W. H— was born ten months after the previous child, who is strong and healthy. Mother contracted gonorrhœa from father two months before birth of W. H—. No family history of tuberculosis.

Previous history.—Very small at birth; weight not recorded. Ophthalmia neonatorum. Child always delicate. Easily caught cold; has had cough on and off since birth; severe attack of bronchitis two months before admission.

Feeding.—Breast-fed four or five months; then Nestle's milk and barley water; then goat's milk, and finally Glaxo. Solid food given at 9 to 10 months. Recently before admission had cow's milk, gravy, potatoes, cream, eggs, bread and milk; altogether a very liberal dietary.

Patient never had diarrhœa or vomiting; bowels have been fairly regular. Motions normal.

At about the age of 6 months mother noticed the back was becoming greatly curved, the curvature increasing. The child was not nursed much, but was allowed to lie at full length on her back in a perambulator, which was long enough for the purpose. At one year the child began to say a few words and to make attempts at taking steps if held, but has never stood alone. The legs have always appeared "strange."

Condition on admission.—Child markedly anæmic and flabby, lies absolutely motionless with legs fully extended, makes no effort to move, and cannot be made to turn over or use legs by any ordinary method of stimulation. Patient has been kept on her back for some months owing to spinal curvature.

The bones show rickety changes in marked degree. Fontanelle not closed, bossing of frontals, marked Harrison's groove, marked curvature of humerus and femur; some bulging of the temporal bones just above the ear; marked kyphosis in lower dorsal and lumbar region with considerable prominence of spinous processes.

All epiphyses of long bones greatly enlarged and well-defined rickety rosary. There was no tenderness of spine, no rigidity, and apparently no pain in jarring.

Heart small and flabby in action. Abdomen pot-bellied. Lungs, no obvious physical signs.

Pathological report.—Urine acid. Small deposit on centrifugalising, consisting of starch granular and bladder epithelium, numerous Gram negative mobile bacilli. No pus. *Blood:* Red blood corpuscles, 3,640,000 per c.mm.; white blood corpuscles, 12,400 per c.mm.; hæmoglobin, 40 per cent.; colour index, 55. Differential count: Lymphocytes, 62 per cent.; polymorphs, 37 per cent.; eosinophils, 1 per cent. No alteration in size or shape of red blood corpuscles. No nucleated forms.

Wassermann test negative.

X-ray report.—Spine: All bones appear poorly mineralised. No indication of tuberculous deposits in spinal vertebræ. All epiphysial lines irregular.

Progress of the case in hospital.—The general condition of the child shows enormous improvement since admission. The temperature, however, has never been steady, but has fluctuated daily between 101° F. and 98° F.

The pulse fluctuates—rate between 116 and 130.

The weight on admission was 1 st. 3 lb. 14 oz., and is now (May the 20th) 1 st. 1 lb. 12 oz. Movements are much more active, and the child can now (three months after admission) sit up by herself and stand up by holding on to the head of the bed. The colour of the child has completely altered, and she no longer appears anæmic.

Since admission there has been one attack of rather severe bronchitis, lasting a few days with high temperature, due to an infection (influenza) which attacked most of the children in the same ward.

The general course of the complaint suggests that in addition to the very marked symptoms of rickets there is an underlying tuberculous process. But the fact that this serious complication has not interfered with the general improvement of the typical rickety symptoms only strengthens the view of the pathogenesis of rickets which I have sketched.

The treatment since admission has been based on the assumption that the marked and typical rachitic symptoms have been caused by a relative excess of food which has called into play certain protective mechanisms, which have brought about a short-circuiting of the oxidation processes and the production of an acidosis. The acidosis has led to an anæmia, and the organism has attempted to counteract this by compensatory over-activity of the hæmogenetic centres in the long bones. Hence the great enlargement of the epiphyses of the long bones.

The acid toxæmia has further led to the paresis of muscles, the general debility and the demineralisation of the bones. The kyphosis is probably postural. Previous to admission the child had been treated by liberality of diet and enforced rest in the recumbent position on the assumption that the spinal curvature was of a tuberculous character, and hence all the conditions were provided for establishing a "vicious" circle of relative over-feeding. Since admission the treatment has been to create a demand for food by massage, resistance exercises, cold douches, and, as far as conditions in hospital allow, by open-air treatment.

The acidosis has been specifically treated by the exhibition of large doses of alkalies, but the alkali tolerance has been very high, and the urine has rarely been alkaline in spite of the alkali administration. The presence of the Gram negative mobile bacilli in the urine may be explained by its acid reaction.

To compensate for the anæmia carbonate of iron has been given, and phosphorated cod liver oil (1 in 10,000) has been supplied with the view of building up the damaged nervous system. The diet has been simply of milk, restricted in quantity.

The general result has been that although the child has lost weight owing to the muscular exercise and loss of fat, the general condition has improved immensely, the epiphyses being noticeably smaller.

I cannot help thinking that the general improvement of the rickety symptoms under this line of treatment, in spite of some underlying condition of an infective and probably tuberculous character, proves that the hypothesis is correct, and that the pathogenesis of the rickety condition, in this case at least, and probably in all cases, is due to causes which may be explained in accordance with the views I have ventured to express.

A CASE OF CONGENITAL URTICARIA PIGMENTOSA, WITH REMARKS ON ÆTIOLOGY.*

By George Pernet, M.D.,

Dermatologist to the West London Hospital and Lecturer on Dermatology, West London Post-Graduate College.

The case was that of a male infant, aged 7 months, first seen by me in the West London Hospital Skin Department on April the 14th, 1916. Scattered about the head, body, and limbs were numerous characteristic pigmented circular and oval lesions, which were flattish and slightly raised above the level of the skin. The distribution is well brought out in the front view of the child (v. photograph). It will be noticed the long axes of the oval pigmented areas are mainly transversely arranged, that is in the lines of cleavage, a fact I find noted in another case observed by me. On stretching the skin over the lesions the yellow tint was well brought out, and on stroking them with the end of the nail, with some pressure, a wheal was produced, a characteristic of the complaint.

* The case was shown at the Dermatological Section of the Royal Society of Medicine on May the 18th, 1916.

Urticaria factitia was also present, as is the rule. The itching was very great, and kept the child and its parents awake at night.

The mother was not suckling the infant, which was being reared artificially, the dietary including groats. The infant appeared quite healthy, but the motions were very dark. The only other thing I

Congenital urticaria pigmentosa.

noticed was just a suspicion of congenital syphilis about the look of the bridge of the nose and the forehead, the former being depressed and the latter somewhat bossy. The Wassermann test was negative in the case of the child and faintly positive in the mother.

Besnier, in his valuable commentaries in the French edition of Kaposi's 'Diseases of the Skin,' points out that adenopathies are always present in these cases. But in this instance, apart from a

very slight amount of adenitis about the groins, they were not present. This may be accounted for by the short duration of the disease in this case. After a long-standing urticaria pigmentosa, with itching and scratching, the glands would have a tendency to enlarge on that account.

A liq. mag. carb. and sod. bicarb. mixture and a lotion of sanitas (1 in 8) were ordered. This, and no doubt leaving the groats out of the dietary, led to much improvement in the itching and the restfulness of the nights all round.

The congenital nature of the condition has been emphasised in the title, for the complaint is usually recorded as manifesting itself in the early months of life. Looking up my notes, I can put my hands on three cases without going far afield. In these the disease first appeared at the age of three weeks in two cases and of six weeks in a third. In the present case, the grandmother, who was present at the confinement, noticed the dark spots at the time of birth. Colcott Fox* states the disease is rarely congenital. In passing I may say there was no suggestion of maternal impressions or longings, such as seeing a leopard or dreaming of a leopard's skin.

The histological appearances in urticaria pigmentosa are peculiar, and may be said to be pathognomonic, viz. numerous mast-cells *arranged in columns and rows*. Mast-cells are found in other morbid conditions of the skin, but not in the same numbers nor with the arrangement just insisted on. Whether these mast-cells are identical with those described by Ehrlich in the blood is doubtful, especially since the investigations carried out by Gilchrist, of Baltimore. The mast-cells in urticaria pigmentosa appear to be rather degenerated connective-tissue cells. Howbeit, the arrangement in rows and columns is *sui generis*.

As to the name, it has been doubted if the condition was essentially a true urticaria at all, and some writers consider the label xanthelasmoidea (Tilbury Fox) better than that of urticaria pigmentosa (first so-named by Sangster). It may be mentioned here that the condition was originally observed and recorded by Nettleship (1869), who described his case as one of "chronic urticaria leaving brown discolorations behind and of two years' duration." On the other hand, the urticarial manifestations may only be associated with the formation of the mast-cell infiltrations. But how these arise is unknown, unless we fall back on the refuge of the destitute, viz. an intoxication, auto- or otherwise, or intoxications of various kinds, which does not take us very far, ætiologically speaking.

* Allbutt and Rolleston, 'System of Medicine,' vol. ix, p. 233.

That some special factor or factors must be at work, in the blood or in the skin, or in both, must necessarily be considered, seeing how uncommon the condition is. I have not seen a case, with the exception of this one, during the last three years in my clinic, although a large number of infants and children pass through my hands.

As to the possibility of syphilis playing a part, that is a point that would have to be worked out specially. But even if we were dealing with an undoubtedly congenitally syphilitic infant presenting urticaria pigmentosa, it might well be merely coincidence. Yet we know that in all morbid conditions of obscure origin, the possibility of syphilis as an ætiological factor lurking in the background must be borne in mind. Without desiring to force analogies, a fallacious procedure as we all know, I may yet be allowed to refer to another rare disease, viz. retinitis pigmentosa, which I believe ophthalmologists are inclined to look upon as of syphilitic origin. I am not insisting on this disease on account of the qualifying adjective, *pigmentosa*, common to both, for the pigmentation arises from a totally different histological substratum apparently in each. No, my point is that in both cases we are dealing with rare conditions, though congenital syphilis is common enough. I am trying to meet the possible objection, a usual one, that because congenital syphilis is of everyday experience, therefore urticaria pigmentosa ought to be common too, which does not follow at all, although the general rule is in favour of the objection. But in these instances we may be dealing with an exception.

The Wassermann test was, as I have said, faintly positive in the mother and negative in the child. We know that in congenital syphilis the Wassermann test is not necessarily positive.

In conclusion, as to ætiology, I do not assert that urticaria pigmentosa is of syphilitic origin, but I throw out the suggestion that it is a point to consider.

Royal Society of Medicine.

DERMATOLOGICAL SECTION.

May the 18th, 1916.

Parakeratosis variegata.—Dr. E. G. Graham Little.—The patient was a girl, aged 18 years, one of three children, and the sole member of the family with any skin disease. She had showed no signs until eight

years previously, when a vivid red rash appeared quite acutely one night on the legs and arms, and was ascribed to her having bathed in the sea when she was hot. The disease spread continuously but slowly until within about two years the entire surface was affected with the exception of the nails. Two years later, at the age of 13, she began to have epileptic fits, which had persisted. There was singularly little interference with the general health.

At the present time the eruption was very nearly universal, in the form of a fine branny lichenoid desquamation with points of accentuated redness as compared with the bluish-red general tone, these points constituting a very indefinite maculo-papule. Over the extensor surfaces of the joints there was much thickening, rather like that of pityriasis, but there was nowhere any resemblance to a follicular papule. The colour of the eruption varied, and at times assumed a vivid blotchy redness like measles, for which it had been mistaken at the onset. Silvery fine scales were freely shed. No treatment had made the smallest impression on the disease.

Lichen scrofulosorum.—Dr. Graham Little also showed a girl, aged 13 years, who had been treated for two years with small doses of tuberculin in the form of bacillary emulsion for scrofulodermia of the neck of five years' duration, resulting from a tuberculous glandular abscess. Lichen scrofulosorum had recently appeared suddenly in the form of a large number of patches of grouped follicular papules, the patches forming oblong or circinate shapes from 1 in. to 4 in. in diameter, and distributed on the back and front of the trunk between the navel and the nipples. The papules were of a vivid red colour, and at the periphery were somewhat more salient and more vivid in colour. The patches had persisted with little change except that there was a slow but progressive fading of colour, probably due to the omission of the tuberculin injections.

Acute Disseminated Tuberculosis cutis.—Dr. Graham Little also showed a male infant, aged 7 months, in whom the eruption had come out quite acutely about a fortnight before. The whole of the skin from the buttocks to the feet was closely covered with an eruption of small, deep bluish nodular lesions varying in size from $\frac{1}{16}$ in. to $\frac{1}{4}$ in. Above the level of the pubes the skin was unaffected. The neck glands had become enlarged at the age of 2 months, and the mastoid had been operated on for tuberculous disease at the age of 3 months. After the removal of about twelve caseous glands, in which bacilli were easily found, the eruption faded considerably.

Congenital Urticaria pigmentosa.—Dr. George Pernet. (See p. 301.)

Société Nationale de Chirurgie de Paris.

A PRIZE OF 50,000 FRANCS FOR THE BEST MECHANICAL HAND APPARATUS.

A generous donor, who wishes to remain anonymous, has offered to the Société Nationale de Chirurgie a prize of 50,000 francs, to be handed over to "the maker of the mechanical apparatus supplying the place of the hand best. All competitors must belong to allied or neutral nations. They are

to present to the Society mutilated men who have been using their apparatus for at least six months. The Société de Chirurgie will experiment each apparatus on mutilated men for the length of time it thinks fit. The apparatus rewarded is to remain the property of its inventor. The competition will be closed two years after the end of the war."

MM. Faure, Kirmisson, Quenu, Rieffel, and Rochard, who form the Committee elected by the Société de Chirurgie, inform the public of the conditions of the competition as stated by the donor, and beg any person wishing to compete to send his essay and apparatus to M. le Secrétaire Général de la Société Nationale de Chirurgie, 12, rue de Seine, Paris.

Abstracts from Current Literature.

Diseases of the New-born.

The secretion of bile in icterus neonatorum (*'Amer. Journ. Dis. Child.,'* 1916, xi, *p.* 295).—**A. F. Hess** refers to his previous paper (*vide* British Journal of Children's Diseases, 1912, ix, *p.* 414), the conclusions of which have been confirmed by his subsequent investigations. He found that the amount of bile-flow during the first twelve to thirty-six hours of life was very scanty, and although it then increased in volume, it was subject to wide variations. In general it was marked when jaundice was marked. J. D. Rolleston.

Severe jaundice in the new born a cause of cerebral diplegia (*'Amer. Journ. Med. Sci.,'* 1915, cxlix, *p.* 345).—**W. G. Spiller** raises the question of the possible relationship between severe icterus neonatorum and cerebral diplegia. In several ways there is the possibility of such a relationship. The arrested growth of the brain might be due to the jaundice or to hæmorrhage resulting from the jaundice; or on the other hand both the cerebral condition and the jaundice might be the result of some common factor. Some of the cases quoted by the author from his own experience and from the literature are suggestive, and one or two are very similar to the case of possible "kernikterus" reported in this country by L. G. Guthrie (*'Proc. Roy. Soc. Med. (Child. Sect.),'* 1914, vii, *p.* 86). Reginald Miller.

Severe tetanus neonatorum (*'La Pediatria Española,'* 1916, v, *p.* 60).—**M. G. Alvarez.**—The patient was a male infant, aged 14 days, brought to hospital because it could neither take the breast nor cry. There were marked trismus, nuchal rigidity, and opisthotonus. The temperature was 104·4° F. The forehead was wrinkled, the eyes staring, and the masseters forcibly contracted. The lips looked as if the child was going to whistle. Clonic contractions were provoked by noise or a bright light. The belly was hard and tense, the lower limbs were over-extended and the upper limbs were forcibly flexed, especially the fingers. Ten c.c. of anti-tetanic serum were given, but the symptoms grew worse and there was a danger of asphyxia from contracture of the respiratory muscles. Subcutaneous injections of oxygen were then given and repeated every other day until

five had been given. About 300 c.c. were used each time. Improvement took place on the second day, and complete recovery resulted.

J. D. Rolleston.

Pneumococcal peritonitis in infants: Possibility of its congenital origin (*'Le Nourrisson,'* 1915, III, *p.* 138).—**G. L. Hallez.**—A female infant, aged 5 weeks, was admitted to hospital for diarrhœa, vomiting, fever, and meteorism, and died the following day. The autopsy showed diffuse peritonitis, from which virulent pneumococci were cultivated. The lungs were normal. The infant had been born slightly before term while the mother was suffering from an acute pulmonary condition, probably lobar pneumonia, and developed the first symptoms of peritonitis shortly after birth. Hallez thinks that the fœtus was probably infected *in utero* by pneumococci in the maternal circulation. There are only five other cases on record of pneumococcal peritonitis below the age of 2 years, including one recorded by H. C. Cameron in the British Journal of Children's Diseases, 1912, IX, *p.* 259, in a male, aged 2 weeks.

J. D. Rolleston.

Multiple deaths in the new born of one family (*'Arch. of Ped.,'* 1916, XXXIII, *p.* 168).—**C. Herrman.**—In a family of eight children seven died of pneumonia, five during the first three weeks, one at 10 weeks, and one at 11 months. Herrman attributes this occurrence to a congenital susceptibility of the respiratory tract. Cultures of the mother's breast showed a marked preponderance of streptococci, but as the blood-cultures of the infant were sterile, there was no definite evidence of the mother having infected it.

J. D. Rolleston.

Meningitis in the new born (*'Arch. of Ped.,'* 1915, XXXII, *p.* 583).—**C. Herrman** has collected eight cases of this rare condition from literature, and records a personal case in a breast-fed infant. Fourteen days after birth it refused to take the breast, became cyanotic, limp, and apathetic, with a temperature of 96° F., and died aged 18 days. The necropsy showed a yellowish-green exudate covering the anterior surface and base of the brain and extending to the cord. The lungs were congested, the other organs were normal. Cultures from the meningeal exudate showed the pneumococcus. The case is interesting as being the only case of pneumococcus meningitis hitherto recorded in the new born, in whom the *B. coli* is the usual causal agent of meningitis; on account of the absence of fever and later the markedly subnormal temperature; and on account of the entire absence of meningeal symptoms.

J. D. Rolleston.

Transitory fever in new-born infants (*'La Pediatria,'* 1915, XXIII, *p.* 768).—**S. Lo Cicero** draws attention to the irregularity of the temperature during the first week of life. There is generally a temperature of 37°–38° C. during and immediately after birth, and then a rapid decline to 35°–36° C., followed by a slow rise to normal, which becomes established after the first week after irregular daily fluctuations. Besides this instability, due to irregular working of the heat-regulating mechanism under the influence of the new external surroundings of the infant, there are in many of them true febrile exacerbations occurring without any objective phenomena or apparent cause. Observations on eighty-three new-born infants, by taking the rectal temperature every three hours during the

first eight days of life, revealed the fact that in about 10 to 20 per cent., between the second and fifth day, a febrile temperature was found, varying from 37·5° to 39° C., rising suddenly, and after a short duration, ending by crisis, without being accompanied by any objective signs, but more marked somnolence or sometimes restlessness. Usually the maximum of temperature coincides with the maximum of loss of body weight. The prognosis is always favourable and no treatment is necessary. The ætiology and pathology are unknown. Probably there are various factors, such as the action of food on the intestinal flora and mucous membrane, abnormal processes of metabolism, the influence of external surroundings, and, above all, the instability of the heat-regulating power of the new-born infant.

VINCENT DICKINSON.

Six cases of hæmorrhage in newly born babies (*'Arch. of Ped.,'* 1915, XXXII, *p.* 838).—**J. B. Manning.**—In all the cases the hæmorrhage was spontaneous, except in one due to circumcision. Blood serum was used in all cases. Two died.

F. R. B. ATKINSON.

Gastro-intestinal hæmorrhage in the new born (*'Dub. Journ. Med. Science,'* 1915, II, *p.* 144).—**Spencer Sheill,** in a paper read at the Royal Academy of Medicine in Ireland, differentiated between ordinary melæna neonatorum and the very fatal type of which so little appears to be known regarding its pathology. Having alluded to what has been published regarding the causation and treatment, he proceeded to relate details of a case under his own care. The child, aged 2 days, passed and vomited so much blood as to be almost moribund. The treatment he adopted was the subcutaneous injection of horse serum—a treatment, so far as he was aware, hitherto untried in such a case. The result was rapid and successful, and the child did well and remained so.

J. ALLAN.

Two cases of intestinal hæmorrhage in the new born (*'Med. Press,'* 1916, I, *p.* 283).—**Mabel M. Crawford** records two cases, one of which terminated fatally. In dealing with such cases we have three objectives: (1) To avoid stimulation of the gastric secretion; (2) to induce coagulation; (3) to support the infant's strength. The most important points in treatment are the immediate administration of horse serum subcutaneously and in large doses, repeated once or twice if necessary, the giving of nothing but water by the mouth, and the maintenance of absolute quiet and warmth. In the fatal case, at the post-mortem examination an ulcer was found on the duodenum very near the pylorus. Seen from the outside, the ulcer showed as a grey circular patch, perhaps 2 mm. in diameter, and around this the blood-vessels were deeply injected. A blood-clot filled this part of the duodenum, and at the place of ulceration the wall was found to be extremely thin. There was neither air nor free fluid in the abdominal cavity, nor were there any adhesions about the ulcer. No other lesion was found.

J. ALLAN.

Syphilis hæmorrhagica neonatorum (*'Hospitalstidende,'* 1916, LIX, *p.* 626).—**A. Pontoppidan** reviews the literature and records two personal fatal cases, characterised by bleeding from the fissures round the mouth and from the umbilicus, and by ecchymoses and petechiæ in the skin of the trunk and extremities.

J. D. ROLLESTON.

Diagnosis of intracranial bleeding in the new born (*'Cleveland Med. Journ.,'* 1915, XIV, *p.* 808).—**H. G. Sloan.**—After difficult or prolonged birth the child should be carefully watched for the next few days for any evidence of cerebral bleeding. Early symptoms of cortical irritation are most valuable in the localisation of the bleeding. Supratentorial hæmorrhage can be differentiated from subtentorial. When symptoms of general increase in intracranial pressure become marked, a decompression operation should be done. The best results are obtained when the pulse-rate is still slow and before the respiration becomes irregular.

J. D. ROLLESTON.

Further experience in the treatment of intracranial hæmorrhage in the new born (*'Boston Med. and Surg. Journ.,'* 1916, CLXXIV, *p.* 947).—**R. M. Green** refers to his previous paper (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1915, XII, *p.* 53), and records two more cases of infratentorial and supratentorial hæmorrhage respectively. The former was treated by lumbar puncture and the latter by incision along the coronal suture, followed by drainage. Both recovered. J. D. ROLLESTON.

Hæmorrhagic disease in the newly born treated by horse serum (*'New York Med. Journ.,'* 1915, CI, *p.* 1274).—**A. Hymanson** relates the case of a male infant, aged 5 days, who began to bleed at the navel and had purpuric spots on the skin. On the eighth day it passed blood from the nose, mouth and bowels, and the purpura increased. Calcium lactate had no effect. Coagulation time of blood eleven minutes. Twelve c.c. of horse serum were injected, with two similar doses next day. Improvement began at once, the bleeding ceased, and the subnormal temperature became normal. Three other cases were similarly treated, and all recovered except one.

J. PORTER PARKINSON.

Citrate transfusion for melæna neonatorum (*'Journ. Amer. Med. Assoc.,'* 1915, LXV, *p.* 2163) —**H. Woltmann** reports a successful case of transfusion into the median basilic vein of 60 c.c. of citrated blood in a baby suffering from melæna neonatorum, after a subcutaneous injection of horse serum had proved of no avail. One hundred c.c. of blood from a vein in the father's arm, and mixed with 10 c.c. of sterile 2 per cent. sodium citrate solution in a sterile container, was employed. T. R. WHIPHAM.

Tubo-ovarian inguinal hernia in a new-born child (*'Rev. Méd. de la Suisse rom.,'* 1916, XXXVI, *p.* 244).—**A. Givel.**—A female infant, aged 6 weeks, presented a pyriform, tender, irreducible swelling in the right labium majus, resembling a hæmatoma. Stools normal, no vomiting nor fever. Operation showed that the tumour was due to a hernia of the Fallopian tube and ovary, which had to be removed, as the torsion of the pedicle had caused an infiltration of blood into the ovary and partial necrosis. Complete recovery followed the operation. J. D. ROLLESTON.

Birth fractures of the humerus (*'Arch. of Ped.,'* 1915, XXXII, *p.* 669).—**E. D. Truesdell** made observations on twenty-four cases in the New York Lying-in Hospital. Seventeen occurred during version with breech extraction, four during simple breech extraction, and three in vertex presentation owing to difficulty in extracting the anterior arm, either from narrowing of the pelvic outlet or from the size of the child. In three the

fracture was double. Seventeen survived. In all but one, in which it was oblique, the fracture was practically transverse, the site of fracture being at or near the centre of the shaft just below the deltoid insertion. Displacement of the fragments was the rule, the prevailing deformity being external angulation. A second variety was lateral displacement. Each fracture was accompanied by a well-marked wrist-drop due to injury to the musculo-spiral nerves. The paper is illustrated by twenty-one skiagrams.

J. D. Rolleston.

Developmentally deficient children twice born to the same couple (*'Amer. Journ. Obst.,'* 1915, lxxii, *p.* 729).—**W. C. Gayler.**—In January, 1914, a woman, aged 23 years, was delivered of a male child at term normal in all respects except for an imperforate anus and complete absence of rectum. It lived twelve days with an artificial anus. In January, 1915, she gave birth to a female anencephalic monster five weeks before term. The father, aged 28 years, was in good health. Neither of the parents and none of the four grandparents had any developmental defects. There was no history of trauma to the mother during either pregnancy, and there was no evidence of alcoholism, syphilis, or tuberculosis in either parent.

J. D. Rolleston.

Congenital oral defect (*'Dub. Journ. Med. Science,'* 1915, ii, *p.* 59).—**Spencer Sheill**, at a meeting of the Royal Academy of Medicine, showed photographs of a child born with an unusual frænum between the upper maxillary bone and the lip. It was not considered of great consequence at the time, and was left; but as the child grew older it grew rapidly, and at the time of development of the teeth he had to cut the frænum, which came level with the tips of the two central incisors. The condition was difficult to explain without going into embryology. He suggested that an epithelial inclusion was pressed out through the two mesial nasal processes.

J. Allan.

Congenital malformation of the intestine (*'Amer. Journ. Dis. Child.,'* 1915, x, *p.* 360).—**L. M. Drennan** and **H. C. Clark.**—A new-born male infant began to vomit as soon as it was put to the breast, and continued vomiting after each feed. The bowels did not move. Laparotomy on the fourth day of life showed a high congenital malformation, causing complete obstruction of the small bowel and other points, indicating stenosis of the small intestine. Operation was therefore abandoned as hopeless, and death occurred a few hours later. Post mortem four complete congenital occlusions of the small bowel were found. The first was 28 cm. from the pyloric ring, and consisted of a thin diaphragm, at the second, which was 15 cm. lower, at the third, which was 10 cm. below the second, and at the fourth, which was 42 cm. below the third, the bowel tapered to a fine solid cord. No other congenital malformations were present except a small foramen ovale.

J. D. Rolleston.

Complete congenital atresia of the ileum (*'Med. Record,'* 1916, lxxxix, *p.* 1039).—**R. H. Fowler.**—Vomiting began about fifteen minutes after birth and continued several times during the night and following day. The abdomen was distended. A diagnosis of complete atresia of the duodenum or hypertrophic pyloric stenosis was made. Operation was performed when the child was 27 hours old. On laparotomy an enormously

distended portion of small intestine, blind and free, presented. Enterostomy was performed, but vomiting continued, and death took place four days after the operation. Post mortem the ileum was found to end in a small blind pouch 22½ in. from the ileo-cæcal valve. There was a defect in the mesentery at a point opposite the situation of the atresia of the ileum. The upper blind pouch ended at a point 33½ in. from the pylorus. It was very much dilated for a distance of 6 in. The ileo-cæcal valve and pyloric sphincter showed no change. The liver, gall-bladder and ducts were normal. There was an accessory spleen. The length of the small intestine was 56 in., *i. e.* 4 ft. 8 in. shorter than normal. J. D. Rolleston.

Imperforate anus and other congenital deformities (*'South African Med. Record,'* 1916, xiv, *p.* 72).—**B. Bernstein.**—A woman who had borne two normal children gave birth to an asphyxiated premature boy (36 weeks) with the following abnormalities: (1) Talipes calcaneus of both feet. (2) More or less complete absence of forearm, which was represented by a short stump in which only one bone could be felt. Probably absence of radius. Attached to the stump was an abnormal hand, in which (3) the thumb and little finger were present, the other three fingers being fused together. (4) Absence of anus. Artificial respiration was stopped when this last deformity was discovered. J. D. Rolleston.

Congenital trophœdema (*'Riv. di Clin. Pediat.,'* 1915, xiii, *p.* 833).—**I. Fini** reports the cases of mother and child suffering from this condition, and discusses the ætiology with reference to congenital œdema and Milroy's disease. With regard to the differential diagnosis between the writer's cases and congenital œdema, she mentions an objective symptom as worthy of special attention, *i. e.* the distribution of the œdema. The favourite site is the lower limbs, but the upper limbs at their distal extremities show also a slight degree of abnormality. Guided by the conception that in the fœtus and at puberty there is a hyperactivity of the genital glands, and especially those with internal secretion, and noting the contemporaneous onset of the œdema which goes on increasing up to a certain point and then is arrested and maintained almost unaltered during life, the writer thought that the relation between the two phenomena might be sought for in a deficient or altered secretion of one of these glands. She gave the mother, therefore, ovarian extract, both fresh and dry. After a month the circumference of the legs had apparently diminished ½ cm., but the woman's weight decreased in the same time by 500 grammes. The child was breast fed and increased regularly in weight; the feet showed no marked change except that the œdema was a trifle softer and had extended slightly.

Vincent Dickinson.

A case of congenital hemihypertrophy (*'Lancet,'* 1915, ii, *p.* 10).—**J. M. Fortescue-Brickdale.**—The patient, aged 9 months, is the only child of healthy parents. There is no history of congenital deformity in any other member of the parents' families. The condition present in the child was first noticed some months after birth, but there appears no reason to suppose that it was not congenital. Beyond a small umbilical hernia, the child presents no sign of disease or deformity other than that about to be described. His mental condition is normal; in fact, he is remarkably bright, active, and intelligent for a child of his age. His skin is normal. There are no nævi or subcutaneous lipomata. The growth of

hair and nails is the same on both sides; he has cut two lower median incisor teeth, which appear to be equally developed, and there is no difference in the temperature of the two sides of the body. The whole of the right side is larger than the left. The disparity is most noticeable in the lower extremities and face. The right ear is larger than the left, the right palpebral fissure is more widely open, and the right side of the tongue and right side of the lower jaw are distinctly larger than the opposite sides. The face has a curiously twisted look, as if the median line, instead of being straight, were the arc of a large circle, the centre of which would fall a yard or two to the left of the head. The parts are well developed, the muscles seem firm, there is no subcutaneous œdema on the larger side. The child, however, seems to use his left hand more readily than the right. The author thinks that the majority of cases of this nature are hemihypertrophies, a hypothesis that seems probable for the following reasons: (1) The distribution of the enlargement may vary from a few digits to the whole of one side. It would be absurd to regard two toes as normal and the rest of the body as atrophied. (2) Cases of enlargement of the opposite limbs are similarly difficult to regard as an atrophic condition of the rest of the body. (3) In several cases the smaller side was more functionally useful than the larger. (4) The few post-mortem examinations do not give any support to the view that the smaller side is atrophic. After a short historical note the author discusses clinical groups and pathogeny of these affections, and gives a bibliography of the more important and recent contributions to this subject. J. Allan.

Pre-maternity nurses and nursing (*'Journ. State Med.,'* 1916, xxiv, *p.* 114).—**J. W. Ballantyne** believes that ante-natal care can hardly fail to have a beneficial effect upon the mortality in children of a year old or under; and the morbidity also which prevails so largely in these early months of life would be greatly lessened by the improvement of the health and hygiene of the expectant mother. He gives an interesting account of the progressive development of the work in the pre-maternity ward in the Edinburgh Royal Maternity Hospital, and he further shows how the good work in this connection might be extended by dealing with extern cases. Nurses should take note not only of the parturient and puerperal patients upon whom they are actually in attendance, but also of prospective patients with whom they come in touch either on their direct invitation or by hearing of them through their patients. The author has prepared inquiry cards which contain the important matters to be discovered during these pre-maternity visits. The card contains spaces for the answers to queries respecting the name, age, address, and usual occupation of patient, her previous pregnancies, labours, and babies, with the mode of feeding them which was employed; her state during the present pregnancy and the expected date of her confinement, and, very important, the presence or absence of persistent headache, vomiting, or constipation, of swelling of the face and hands, of discharge, especially red in colour, from the vagina, of failure of eyesight, and of any other thing which has been specially troubling the patient, or which may by its unusual character attract the attention of the nurse. The rest of the card contains reminders to the pre-maternity nurse to give instruction and advice, if required, upon diet, fresh air (open window), avoidance of strains (lifting, reaching), clothing preparation for the confinement and the baby, care of the breasts and teeth, and upon the importance of reporting herself to the hospital, or of asking

for another visit from the nurse upon the occurrence of any one of the unusual symptoms or signs of pregnancy, *e. g.* red discharge with pain in the back, too much morning sickness, foul discharge, etc. By an organised working on the lines suggested much might be done in the cause of child welfare, for by watching over and caring for the mother in her pregnancies one is attending to the welfare of the child at the time when it needs it.

J. Allan.

Diseases of Metabolism.

Mild diabetes in children (*'Amer. Journ. Med. Sci.,'* 1916, cli, *p.* 40).—**D. Riesman** states that there is a mild type of diabetes in childhood and adolescence which tends to affect several members of the same family. The glycosuria is usually moderate and under 1 per cent., although nervous excitement and other factors may increase it. Other diabetic symptoms are often slight and may be absent. The disease is not progressive and may end in apparent recovery; in its general features it corresponds to the so-called renal diabetes. Riesman records four cases, three of which were in members of one family, aged from 16 to 22 years, and one in a girl, aged 14 years.

J. D. Rolleston.

Diabetes and suckling (*'Riv. di Clin. Pediat.,'* 1915, xiii, *p.* 428).—**G. Guidi**, of the Children's Hospital at Florence, contributes a short but interesting paper on this subject. It does not deal with alimentary glycosuria preceding gestation, but with a special glycosuria which makes its appearance insidiously as gestation advances, and is characterised by the fact that, in contradistinction to the common glycosurias in pregnant or non-pregnant women, it is entirely independent on any kind of food and never induces hyperglycæmia. It is possible that this kind of gravid glycosuria is of renal origin and has some relation to abnormal permeability of the kidney to sugar, a renal anomaly due to gestation and experimentally confirmed by Pollak, who was able to reproduce this peculiar condition of the kidney by preparations of uranium and chromium. The author's theory is that certain elements of the genital organs, and more especially certain interstitial cells of the ovary which form an internal secretion—endocrinic cells—have some relation with the onset of glycosuria during pregnancy. The glycosuria ceases during the puerperium when these elements of the genital organs, owing to uterine involution, cease their excessive activity caused by gestation and revert to their normal function. In confirmation of this theory is the fact that the glycosuria seems to persist beyond the puerperium if the woman suckles, and this is doubtless because, as shown by Brugnatelli, in the interstitial tissue of the mammæ also there exist elements which present a marked analogy with the above-mentioned interstitial cells of the ovary. Hence it is logical to conclude that the products of secretion of these tissues, by way of proliferation and hyperfunction during the advanced period of pregnancy, act on the kidney, the organ of elimination, in the same way that uranium and chromium act, inducing an abnormal permeability to sugar, and in this way causing glycosuria. From the cases observed by him the author is of opinion that suckling is contra-indicated under such circumstances, as it is likely to cause severe intoxication in the child, with fever, vomiting, tetany, and even a fatal issue. Gravid glycosuria causes no harm to the fœtus as long

as it is in the uterus, but it will feel its toxic effects as soon as the mother's milk conveys to the infant the toxins developed in the mother by the changes induced by disturbed renal function, and puts them into the circulation of an infant already hypersensitive from sojourn in the womb of a mother who is toxic although not hyperglycæmic.

VINCENT DICKINSON.

A case of diabetes ('*Lancet*,' 1916, I, *p.* 1115).—**W. G. Smith** records a case in a boy, aged 7 years, and makes some interesting comments, especially in regard to acidosis. J. ALLAN.

Acidosis in children ('*Boston Med. and Surg. Journ.*,' 1915, CLXXII, *p.* 747).—**A. Howard** describes sixty-four cases—thirty in males and thirty-four in females. The age varied from 2 days to 12 years. The cases under 2 years were as frequent as older patients. Acidosis, with recurrent vomiting and without any predisposing cause, is a very rare condition. The predisposing causes are acute infections, *e.g.* tonsillitis, bronchitis, otitis media, and infectious diarrhœa, and other states such as intestinal intoxication and stasis, burns, and etherisation. The severity of the acidosis in no way depends upon the gravity of the prodromal condition. Constipation is the rule. Twenty-two of the sixty-four gave histories of repeated attacks. The disease is ushered in by restlessness and irritability, the lips and mouth become dry, there is abnormal thirst, the respirations are rapid, and acetone appears in the breath and urine. Nausea and vomiting ensue. The irritability gives place to drowsiness, which may deepen into coma. Vomiting occurred in fifty-one of the sixty-four cases, and was most marked in the early stages of the illness. Three died. Treatment consisted in rest in bed, abstention from food, and glucose and bicarbonate of soda alternately, preferably by the mouth. Food should be started with caution, and a barley lactose solution is the first step to a fuller diet. CHRISTOPHER ROLLESTON.

Acidosis with special reference to that occurring in diseases of children ('*Bull. Johns Hopkins Hosp.*,' 1916, XXVII, *p.* 63).—**J. Howland** and **W. McKim Marriott**.—Acidosis is not uncommon in infancy and childhood. It is especially frequent in the severe diarrhœas of infancy, but may appear in a variety of diseases, and sometimes, apparently, alone. In older children it is recognised by the character of the respiration (hyperpnœa) and laboratory tests. In infants who are irritable, restless, and crying, it is difficult to say if hyperpnœa is present. Older children react promptly to alkali therapy, but infants usually die, though it may be possible to stop the clinical and laboratory evidence of acidosis. Alkalies may be given by mouth, by rectum, subcutaneously, or intravenously. A 4 per cent. solution of bicarbonate of soda is usually employed intravenously, and a 2 per cent. solution subcutaneously. J. D. ROLLESTON.

Acid intoxication in infancy and childhood ('*Boston Med. and Surg. Journ.*,' 1916, CLXXIV, *p.* 568).—**J. L. Morse.**—The most characteristic symptom, apart from the presence of considerable quantities of acetone bodies in the urine, is hyperpnœa without cyanosis. The rate of respiration is increased, both inspiration and expiration are prolonged, the normal relation between the two being observed. Another symptom is diminution in the output of urine. Vomiting is very common and diarrhœa is not

infrequent, but there is nothing characteristic about either, and in many cases there is constipation. The breath has not infrequently a peculiar aromatic odour, but this is not always present and is decidedly difficult to recognise. Acid intoxication has been found in recurrent or cyclical vomiting, in the severe diarrhœa of infancy and early childhood, and in the course of or following various infectious diseases. Degenerative changes have been found in the parenchymatous organs, but nothing pathognomonic. Morse records five illustrative cases in infants aged from thirteen to twenty-eight months. J. D. Rolleston.

Acidosis with auto-intoxication in infants and children (*'Amer. Journ. Dis. Child.,'* 1915, ix, *p.* 28).—**C. R. Metcalfe** reports on 100 cases which occurred in apparently epidemic form at Concord in the course of $2\frac{1}{2}$ months. The ages ranged from 1 to 13 years, but the great majority—eighty—were between 2 and 8 years. Not more than a quarter of the children were neurotic. In twenty-one, adenoids were noted. Six had both adenoids and enlarged tonsils. All degrees of severity were seen. Nine died. The average duration of the fatal cases was thirty-two hours. An autopsy in one case showed fatty degeneration of the liver and congestion of the brain. The epidemic was one of first attacks, eighty-four were ill with acidosis for the first time, eleven cases were recurrent. Considerable quantities of acetone were present in the urine in every case. In only forty-five was the test for diacetic acid made and proved positive in fifteen. Oxybutyric acid was found only once. Treatment consisted in administration of alkalies by mouth or rectum, catharsis, rectal irrigation, and regulation of diet. J. D. Rolleston.

The elimination of uric acid in the infant (*'Riv. di Clin. Pediat.,'* 1915, xiii, *p.* 662).—**C. Francioni** gives details of twenty-six cases to show that increase in the elimination of uric acid of endogenous origin in the infant is due to different factors, such as the presence of disturbances of nutrition on the one hand, and of constitutional anomalies (neuroarthritic or eczematous diathesis) on the other. The increased products of the first factor are usually much more noticeable and constant than those due to the second. In the breast-fed infant, in order to determine whether in any given case the increased elimination of endogenous uric acid is due to the one or the other cause, an estimation of the phosphoric elimination has to be made in order to ascertain with sufficient exactness if the functions of nutrition are taking place normally. In the artificially reared infant this method of investigation is of no value, since phosphoric elimination is always greatly increased, and on the other hand uric acid elimination is too much under the influence of the nutritional function to be taken as an index of the diasthetic condition. Vincent Dickinson.

Excretion of magnesium sulphate injected subcutaneously (*'Amer. Journ. Dis. Child.,'* 1915, ix, *p.* 318).—**A. M. Courtney** and **H. L. Fales** derived great benefit from the use of magnesium sulphate in the convulsions of tetany in infants. In three cases they studied the magnesium and sulphuric acid metabolism on the days following injection. They found that there was no danger of a cumulative effect if the dose (4-10 c.c. of a solution of 8 grm. anhydrous $MgSO_4$ per 100 c.c.) was not repeated more than once in twenty-four hours. The injected SO_4 was immediately and completely excreted in the urine. J. D. Rolleston.

The amount of lipase in the organs (*'La Pediatria,'* 1915, XXIII, *p.* 625).—**A. Clerici**, from a series of experiments conducted on adult and young animals of varying ages, finds that the liver is the richest of all the organs in lipase, both in the young and in adults. The organs of the young have less lipase than the same organs in adults. This difference is especially noticeable in the case of the kidney, the renal extract of young and very young animals invariably contain less of the ferment than renal extract in adults. VINCENT DICKINSON.

Ophthalmology.

The blind child (*'New York State Journ. Med.,'* 1916, XVI, *p.* 23).—**F. P. Lewis** emphasises three things: (1) The necessity of recognising the fact that the child is blind, or has defective sight, at the earliest age possible; (2) the manner in which the brain is developed through the training of the remaining special senses in the absence of sight; (3) the existence of that unusual condition, more highly developed in the blind than in those who see, and what is sometimes termed the sixth sense, or touch at a distance, but which consists in the recognition of the nearness of any material body having a sufficiently large surface area, by the perception of its approach through some sense other than that of conscious sight, hearing, or personal contact. The author discusses how to determine whether the child is blind, how the blind baby should be dealt with, and how the blind child should be educated. J. ALLAN.

Ophthalmia neonatorum (*'Boston Med. and Surg. Journ.,'* 1916, CLXXIV, *p.* 745).—**G. H. Thompson.**—The State of Massachusetts provides the following preventive measures: (1) Notification of birth in forty-eight hours. (2) Use of an approved preventive in every lying-in hospital. (3) Free distribution of prophylactic. (4) Immediate reporting (six hours) by physician, nurse, or hospital, of ophthalmic cases. (5) Lying-in hospitals must report to State Department of Health on cured cases leaving hospital. (6) Authorisation of boards of health to deal with such cases. (7) State officers must advise with local boards of health and follow up cases reported. The result of these measures has been a considerable reduction in the number of cases. J. D. ROLLESTON.

Affection of the lacrymal ducts in the new born (*'Thèses de Paris,'* 1915-16, *No.* 92).—**Z. Hourmouziadès.**—Out of a total of 100,000 cases of eye disease treated at the Fondation Adolphe de Rothschild from 1912–1916 there were 250 cases of congenital affections of the lacrymal ducts. The ages of the patients ranged from 11 days to 4 years. One hundred and twenty-one were males, 129 females. In 86 the left eye, in 78 the right eye, and in 86 both eyes were affected. The most frequent condition observed was simple lacrymation with or without catarrhal conjunctivitis, and next most frequently purulent or muco-purulent dacryocystitis with or without conjunctivitis. The phenomena are due to an imperforate state of the nasal orifice at birth. The prognosis as a rule is favourable. The best treatment consists in methodical massage in the region of the lacrymal sac, combined with daily instillations of a 1 per cent. solution of silver nitrate. If a cure is not obtained by this means within a fortnight, catheterisation of the duct, followed by injections, will be required. J. D. ROLLESTON.

Blue coloration of the sclerotics and abnormal fragility of the bones (*'Arch. f. Chir.,'* 1915, cvii, *p.* 279).—**W. Hofmann** reviews the literature and records three personal cases: (1) Boy, with definite blue coloration of both sclerotics. At 6 years he broke his right leg, at 12 years the left clavicle, at 13 years the right metatarsus, and at 14 years the right ulna. (2) Man, aged 24 years, with fracture of the right patella from fall on knee. Deep blue coloration of both sclerotics. Fracture of left leg at 1 year old. His mother also has blue eyes, but not his sisters. (3) Man, aged 31 years, with deep blue-grey sclerotics. Fracture of right elbow when 1 year old. In 1913 he broke his left little finger and shortly afterwards his right ring finger. His brother, who also has blue sclerotics, has broken his left leg seven times and his right leg three times. Hofmann attributes the anomaly to a congenital weakness of the tissue of the mesenchyma, and regards it as a stigma of degeneration.

J. D. Rolleston.

A case of amaurotic family idiocy in one of twins (*'Arch. of Ped.,'* 1915, xxxii, *p.* 902).—**C. Herrman.**—The mother had been married nineteen years and had been pregnant six times previously. Both infants developed normally during the first five months; after that the patient paid less attention to his surroundings, was less active, and at 8 months was not able to hold his head up properly, whereas his twin sister could sit up alone. His first teeth appeared at 14 months, his sister's at 12 months. Examination of the fundus at the age of 20 months showed the characteristic cherry-red spot. The Wassermann and von Pirquet tests were negative.

J. D. Rolleston.

The cause of the ophthalmoscopic appearances in amaurotic family idiocy (*'Lancet,'* 1916, i, *p.* 352).—**G. Coats**, after dealing with the contribution by F. E. Batten and S. Mayou on "Family Cerebral Degeneration with Macular Change" (*vide* British Journal of Children's Diseases, 1915, xii, *p.* 158), states that for a histological study of the retina the material must be absolutely fresh, as post-mortem changes begin two hours after death; fixation must be perfect (Zenker's solution being the best), and the method of cutting must be suitable. He considered that in the retina, apart from the occurrence of œdema, the macular changes show marked differences in the two diseases, and these were enumerated. He repudiates the view which has been ascribed to him that the ophthalmoscopic appearances in amaurotic family idiocy are due to coagulation necrosis. Though the particular form of chromolysis, vacuolisation, etc., which characterise family cerebral degeneration closely simulates that which is seen in amaurotic family idiocy, the underlying cause may be different; and the race selectiveness of amaurotic family idiocy should cause hesitation in identifying it with a disease which is not race selective.

J. Allan.

The cranial deformity of oxycephaly (*'Amer. Journ. Med. Sci.,'* 1916, cli, *p.* 840).—**W. Sharpe.**—These cases are fairly common and are usually brought first to the ophthalmologist for failing vision due to secondary optic atrophy of varying degree. Sharpe regards the condition as primarily due to an early meningeal irritation with resulting premature closing of the sutures of the base and posterior portion of the skull. As the brain enlarges it tends to protrude through the anterior fontanelle, thus producing the characteristic deformity, and as the result of the increased intracranial

pressure a choked disk with secondary optic atrophy occurs. Sharpe records four illustrative cases in children, aged from 4–15 years, in which subtemporal decompression was performed with good results.

J. D. Rolleston.

Trachoma and follicular conjunctivitis (*'New York Med. Journ.,'* 1915, cii, *p.* 1003).—**M. B. Beals.**—The usual routine treatment by bluestone, tannin, glycerids, argyrol, etc., has shown poor results in thousands of New York children. The author recommends the following treatment, which he calls " the intense bichloride rub " : Instill 1 drop of 10 per cent. solution of cocaine in each eye at intervals of five minutes for three applications, wait fifteen minutes ; fix a pledget of cotton wool the size of a pencil on a steel rod, dip this in 1 in 1000 perchloride of mercury solution, and having raised the lid from the eyeball, pass the wool as far up as possible, release the eyelid and pass the wool slowly from side to side, keeping the probe well away from the eyeball. Do the same with the lower conjunctiva. This should be repeated in five to seven days. If there be pain, a cold application may be used. One in 5000 bichloride solution should be instilled three times a day between the rubs. A feature of this treatment is that no scar tissue is formed. J. Porter Parkinson.

Chancre of the bulbar conjunctiva in a child (*'Journ. de Méd. de Bordeaux,'* 1916, lxxxvii, *p.* 170).—**H. Fromaget.**—A boy, aged 10 years, presented an oblong ulcer of the right bulbar conjunctiva, 10 mm. long by 4 mm. wide, covered by a diphtheroid deposit. There was some pericorneal chemosis, but hardly any discharge, and no pain. In the right parotid region there was a mass of enlarged glands. There was some mottling of the chest and abdomen. Wassermann's reaction was positive. Rapid disappearance of the lesion followed intravenous injections of neo-salvarsan, but prolonged courses of treatment by biniodide of mercury pills was ordered (*cf.* British Journal of Children's Diseases, 1913, x, *p.* 522).

J. D. Rolleston.

Meningococcal uveitis (*'Thèses de Paris,'* 1915–16, *No.* 17). — **F. Gaudin.**—Uveitis occurs in from 4 to 6 per cent. of cases of meningococcal meningitis. It is almost always unilateral. The lesions may start in the anterior segment, giving rise to acute purulent irido-cyclitis, followed by panophthalmia ; but more frequently the process is subacute and plastic, and almost always entails atrophy of the globe. In other cases the deep parts are affected first, giving rise to suppurative choroiditis or the formation of a pseudo-glioma, or, in very mild cases, to foci of disseminated choroiditis. The affection sometimes ends in a mere diminution of visual acuity, but almost always it leads to blindness, with atrophy of the globe or the formation of a pseudo-glioma. Local serotherapy gives the best results (*vide* British Journal of Children's Diseases, 1916, xiii, *p.* 13). The thesis contains the histories of eleven cases, nine of which occurred in children aged from 7 months to 12 years. J. D. Rolleston.

Ocular complications of cerebro-spinal meningitis (*'Presse Méd.,'* 1916, xxiv, *p.* 8).—**Rohmer** records a case of suppurative iritis in a child, aged 10 years, suffering from cerebro-spinal meningitis. In addition to treatment by anti-meningococcal serum, two subconjunctival injections were

given consisting of 1½ c.c. of serum taken from a blister on the child's arm. Improvement occurred the next day, and recovery took place.

J. D. Rolleston.

Optic neuritis with cerebellar ataxia in pertussis (*'Deutsch. Zeitschr. f. Newenheilk.,'* 1915, LIII, *p.* 321).—**A. Strümpell.**—A boy, aged 4½ years, developed a typical attack of whooping-cough in the middle of July. On August 12th he complained of his eyes, and on the 13th became quite blind and was unable to stand. The symptoms did not follow an attack of coughing, and the paroxysms had already begun to decline in severity. Ophthalmoscopy showed marked optic neuritis. The ataxia disappeared in about three or four weeks, but it was not till the end of October that the optic neuritis cleared up. Strümpell attributes the condition to encephalitis, as in the case described by von Domarus (*vide* British Journal of Children's Diseases, 1912, IX, *p.* 326).

J. D. Rolleston.

Injury to eye from explosion of golf ball (*'Journ. Amer. Med Assoc.,'* 1916, LXVI, *p.* 1304).—**E. J. Bernstein** reports another case of a severe burn to the eye caused by the bursting of the core of a golf ball. The patient, a girl, aged 12 years, had been using the ball as a toy. The eye recovered, leaving only a slight central leukoma (*vide* British Journal of Children's Diseases, 1913, X, *p.* 521; 1914, XI, *p.* 281).

T. R. Whipham.

Bird-shot removed from the vitreous through the pupil (*'Journ. Amer. Med. Assoc.,'* 1915, LXV, *p.* 1231).—**K. Pischel** reports the case of a boy, aged 12 years, who two days previously had received a shot in his left eye. The cornea and iris were injured and the lens was cataractous. A skiagram showed a shot in the vitreous downwards and inwards. The cataract was removed in the hope that it might be possible to remove the shot later. The patient was sent home, but returned nearly three months after the accident with the history that the day before, while wrestling with another boy, he fell on him and bumped his head against the other boy's, with the result that he felt a sharp pain in the left eye lasting several hours. The shot was to be seen lying in the lowest part of the anterior chamber, whence it was removed under an anæsthetic. A month later vision was considerably impaired, and behind grey bands in the vitreous an apparently detached retina could be seen. T. R. Whipham.

Glioma of the retina (*'Pediatrics,'* 1916, XXVIII, *p.* 193).—**J. H. Hester.**—Glioma is the only tumour known to arise in the retina. It is essentially malignant, and occurs only during infancy and early childhood. In most cases it is probably congenital. It is more frequent in males than in females. There are four stages. In the first the disease is manifested only by a golden-yellow reflex from the pupil of the affected eye, and by diminution or complete loss of vision. The second stage is characterised by increased tension, and the eye becomes irritated and painful. In the third stage the tumour grows outward from the eye, first along the optic nerve and then in other directions, especially through the cornea. The eye at length is transformed into a bleeding mass, which fills the entire orbit. In the fourth stage the tumour spreads to adjacent and remote organs, and death takes place by exhaustion or by extension of the neoplasm to vital

organs. The disease usually lasts several years. Hester records a personal case in a baby boy of 22 months. Enucleation of the eye was performed, but recurrence took place. J. D. Rolleston.

Operative treatment of high myopia (*'Lancet,'* 1915, II, *p.* 1141).—**A. H. Thompson**, at a meeting of the Royal Society of Medicine, Section of Ophthalmology, read a paper on the late results of the operative treatment of high myopia. The degree most suitable for operation was 16 to 22 diopters. In young children, whose myopia was certainly progressive, a limit of 14 D. might well be assigned. With a myopia of more than 22 D. the chance of the eye remaining free from destructive processes was small. His method had been a free needling of the lens capsule, after which it was necessary to watch the patient carefully day by day, and in the event of a rise of tension let out the lens matter by means of a keratome incision through the cornea. Should a secondary membrane form some years after, this must be needled. He did not think the operative treatment of high myopia tended to counteract the increase in the long axis of the eyeball, though obviously the removal of the lens had the effect of diminishing the result of the lengthening of the axis of the eyeball on the refraction by about one half. J. Allan.

Review.

Diseases of Infants and Children. By Henry Dwight Chapin, M.D., and Godfrey Roger Pisek, M.D. Third edition. Royal octavo. Pp. xviii + 578, with 179 illustrations and 12 coloured plates. London: Baillière, Tindall & Cox, 1916. Price 15*s.* net.

The third edition of the 'Diseases of Infants and Children,' by Drs. Chapin and Pisek, of the New York Post-Graduate Medical School and Hospital, presents no new or striking features. Into some 550 pages the authors have set themselves to compress the whole of our knowledge of the disorders of childhood from diarrhœa to paramyoclonus multiplex. Room is even found for sections upon surgical disorders and for the diseases of the skin, ear, and eye. In such narrow limits it is not possible to do more than present a framework, which contains little or no trace of the distinguished authors' own views and predilections. On the whole it can be said with confidence that the space available has been used to the best advantage. The chapters on feeding are the most complete in the book, and contain many useful recipes, although the names of some of the ingredients sound strange to English ears. The coloured prints of the rashes of the exanthemata, with the exception of Plates IX and X, are of little value, and might well be omitted. On the other hand, many of the photographs reproduced have high value as illustrations, and the selection is well made. The authors make no attempt to avoid the use of a commonplace medical jargon. We read of "septic rises of temperature," for example, of a "negative chest" and of "disease conditions." On p. 96 is the statement: "Mensuration of an atropic (*sic*) extremity or muscle groups are made in cases of infantile paralysis." H. C. C.